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Current views on temperature-modulated *R* gene-mediated plant defense responses and tradeoffs between plant growth and immunity

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Elevated ambient temperatures will likely be a key consequence of climate change over the next few decades. Adverse climatic changes could make crop plants more vulnerable to a number of biotic and abiotic stresses, which would have a major impact on worldwide food production in the future. Recent studies have indicated that elevated temperatures directly and/or indirectly affect plant–pathogen interactions. Elevated temperatures alter multiple signal transduction pathways related to stress responses in the host plant. High temperatures can also influence plant pathogenesis, but little is known about the molecular mechanisms associated with such effects. An improved understanding of the molecular genetic mechanisms involved in plant immune responses under elevated temperatures will be essential to mitigate the adverse effects of climate change to ensure future food security. In this review, we discuss recent advances in our understanding of the effects of temperature on resistance (*R*) gene and/or regulators of *R* genes in plant defense responses and summarize current evidence for tradeoffs between plant growth and immunity.

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Introduction

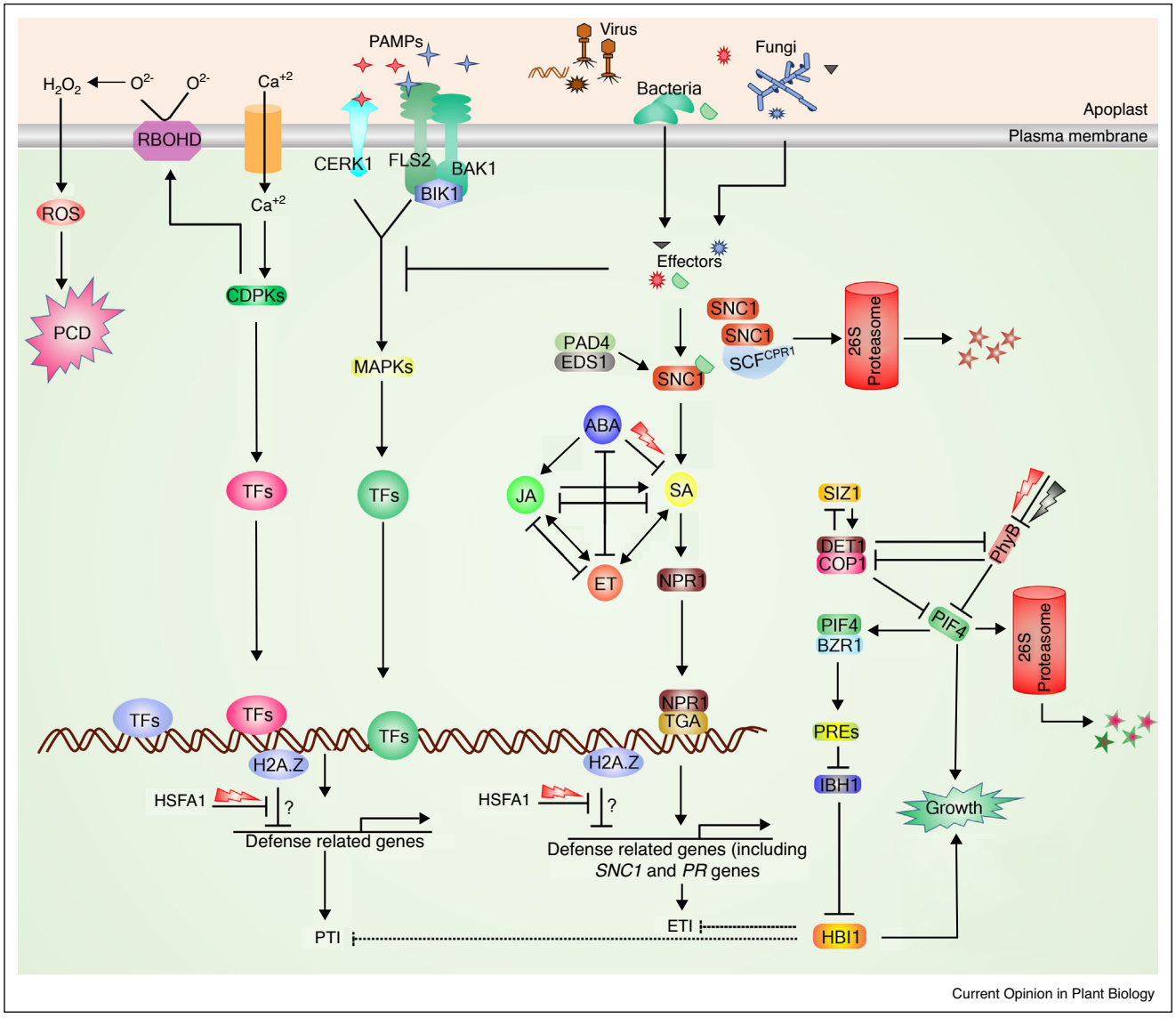
Plants are exposed to diverse pathogens in their natural habitats and have evolved multi-layered defense systems to ward off pathogen attack. Unlike animals, plants do not possess adaptive immunity, instead relying largely on innate immunity. This system utilizes two major classes of receptors: extracellular pattern recognition receptors (PRRs) and intracellular nucleotide-binding leucine-rich repeat (NLR) immune receptors. The detection of pathogen-associated molecular patterns (PAMPs) and pathogen effectors by their cognate immune receptors activates evolutionarily conserved immune responses known as PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI), respectively [1]. Although PTI and ETI employ different receptor recognition mechanisms, both share some common defense signaling events, such as the accumulation of reactive oxygen species (ROS), antimicrobial compounds and defense hormones, transcriptional reprogramming, and the activation of defense-related genes (Figure 1). The abnormal accumulation or activation of immune receptors is deleterious to plant growth and development and, therefore, must be tightly regulated.

Plant defense responses are strongly affected by environmental conditions. Among these, temperature plays a major role, influencing plant–pathogen interactions as well as the timing and severity of disease epidemics. High temperature accelerates the breakdown of plant disease resistance in many plant–pathogen systems, although in some cases, high temperature enhances disease resistance [2]. The expression of several plant resistance (*R*) genes is modulated by temperature (see Ref. [2]), but the crosstalk between NLR signaling and temperature stress responses is not fully understood. Expanding our knowledge of the interplay between plant defense responses and temperature will be crucial for developing effective management strategies to enhance crop resilience to highly variable climatic conditions. In this review, we summarize recent insights into the interplay between temperature and plant immune responses. We also discuss potential mechanisms by which temperature modulates plant defense responses.

Effects of temperature on NLR-mediated immune signaling

Increasing evidence suggests that plant immune responses are strongly affected by temperature. Gain-of-function mutations in NLR genes are implicated in the improper activation and deregulation of these genes, leading to defective growth and temperature-dependent autoimmunity. For instance, in the *Arabidopsis thaliana* protein SNC1 (SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1), the most widely studied NLR protein, a mutation in the linker region between the nucleotide binding domain and the LRR domain enhances the

Figure 1



Schematic model of temperature-mediated defense signaling in plants. Upon perception of PAMPs by PRRs (such as FLS2 [FLAGELLIN SENSITIVE2], EFR [EF-TU RECEPTOR], and CERK1 [CHITIN ELICITOR RECEPTOR KINASE 1]), the basal defense response is activated in plants. Under nonpathogenic conditions, SCF^{CPR1}/proteasome-mediated degradation maintains protein turnover. Under normal growth conditions, SIZ1 activates the ubiquitin E3 ligase activity of COP1 through sumoylation, in turn resulting in the ubiquitination and degradation of SIZ1 and the subsequent inhibition of SNC1 accumulation by PIF4-mediated repression. Inactivation of PhyB at elevated temperature or in dark leads to PIF4-mediated suppression of defense responses mediated by SNC1. PIF4–BZR1 negatively regulates defense responses by promoting the activity of HBI1 through transcriptional regulation of PRE genes. PREs relieves inhibition of HBI1 through direct interaction with IBH1. In addition, elevated temperature induces transcriptional reprogramming, including the rapid replacement of H2A.Z mediated by HSFA1, allowing stress-responsive transcriptional regulators to be induced. SA, JA, and ET are the major hormones involved in plant defense responses, while ABA modulates plant defense responses, either alone or in coordination with the other hormones. Under high temperature stress, ABA inhibits SA biosynthesis and signaling, leading to suppression of defense responses. The red and black lightning symbol indicates elevated temperature and dark conditions, respectively. Blunt and pointed arrows indicate inhibition and activation, respectively. BAK1, BR INSENSITIVE 1 (BRI1)-ASSOCIATED RECEPTOR KINASE 1; BIK1, BOTRYTIS-INDUCED KINASE 1; CDPKs, calcium-dependent protein kinases; MAPKs, mitogen-activated protein kinases; NPR1, NONEXPRESSER OF PR GENE 1; PCD, programmed cell death; RBOHD, RESPIRATORY BURST OXIDASE HOMOLOG B; TFs, transcription factors; TGA, TGACGTCA cis-element-binding protein (TGA) factor.

accumulation of SNC1 and causes a constitutive immune response [3]. However, this immune response is inhibited at elevated temperatures. The inhibited nuclear accumulation of several NLR proteins is associated with the suppression of plant immune responses at elevated temperatures [4,5]. For example, the reduced nuclear accumulation of SNC1 at 28°C is associated with the suppression of the autoimmune phenotypes of the *snc1*

mutant [4]. Similarly, the nuclear accumulation of the tobacco NLR protein, N, conferring resistance to tobacco mosaic virus, is inhibited at 28°C [5]. Loss-of-function mutations in genes encoding negative regulators of plant immune responses are also implicated in temperature-modulated autoimmune immune responses. A number of regulators associated with SNC1-mediated immune responses, including RNA binding proteins, ubiquitin ligases, farnesyltransferase, chaperone complex proteins, and nuclear pore complex proteins have been identified through genetic screening for suppressors and enhancers of *snc1-1* (Table 1). These finding suggest that NLR proteins are regulated by multiple cellular pathways, including transcriptional reprogramming, RNA processing, protein stabilization, and nuclear accumulation. However, the mechanisms underlying the crosstalk between temperature-modulated plant growth and immune responses are not well understood.

Crosstalk between temperature and immune responses

In plants, temperature sensing is associated with fluctuations in membrane fluidity, histone modifications, the activation of protein kinase cascades, and the generation of ROS [6]. Recent evidence highlights the major role of the canonical light receptor Phytochrome B (PhyB) as a thermosensor in Arabidopsis [7,8]. PhyB exists in two reversibly interchangeable states: the biologically active form, Pfr, in red light and the inactive form, Pr, in far-red light. In addition to the light-dependent conversion of Pr to Pfr, temperature-dependent dark reversion switches Pfr into the inactive Pr state.

PhyB in the Pfr state directly interacts with phytochrome interacting factors (PIFs), a class of basic helix-loop-helix (bHLH) transcription factors that regulate several physiological processes in plants [9,10]. Among PIF homologs, the key roles played by PIF4 in high temperature-induced morphogenesis, including the regulation of hypocotyl and petiole elongation responses, the control of auxin-responsive gene expression, and the induction of early flowering are well documented [9–11]. PIF4 functions directly downstream of PhyB-mediated signaling to integrate light and high ambient temperature signals [7–9]. In the dark, PhyB is in the biologically inactive Pr state, while PIFs are highly abundant in the nucleus and repress the transcription of downstream photomorphogenesis-related genes [7,8]. Upon light perception, PhyB switches to the biologically active Pfr state and interacts with PIF4, consequently leading to the phosphorylation, ubiquitination, and proteasomal degradation of PIF4 and hence, the de-repression of PIF4-target gene expression [7,8]. Like light signaling, high temperature leads to the conversion of Pfr into Pr, resulting in the inhibition of PIF4 degradation and thus the promotion of thermomorphogenesis [7,8,12,13].

Table 1

Regulators of SNC1-mediated immune responses in Arabidopsis

Gene	Protein	Function	Mode of regulation	Reference
MOS11	RNA binding protein	mRNA export	Negative	[59]
MOS3	Nucleoporin 96	mRNA export	Negative	[60]
MOS4	Homolog of human BCAS2	Putative function in RNA splicing	Negative	[61]
MOS6	Importin α-3	Nucleo-cytoplasmic protein trafficking	Negative	[62]
MOS7	Homolog of human Nup88	Nuclear protein export	Negative	[63]
MOS8	β-Subunit of farnesyltransferase	Protein farnesylation	Negative	[64]
PAD4	Triacylglycerol lipase	Stabilization of EDS1	Positive	[65,66]
EDS1	Lipase-like proteins	Forms a complex with SAG101 or PAD4	Positive	[66]
BIR1	Receptor-like kinase	Interacts with BAK1	Negative	[67]
BON1	Copine protein	Phospholipid-binding	Negative	[68,69]
BAP1	C2 domain protein	Phospholipid-binding	Negative	[68]
MKP1	Map kinase phosphatase 1	Activation of MPK4	Negative	[70]
SIZ1	SUMO E3 ligase	Protein stability	Negative	[22]
SAUL1	E3 ligase	Protein stability	Positive	[43*]
COP1	RING-type ubiquitin E3 ligase	Protein stability	Negative	[15]
DET1	Nuclear protein	Protein stability	Negative	[15]
PIF4	Phytochrome interacting factor	Transcriptional regulation	Negative	[15]
SRFR1	TPR domain-containing protein	Protein stability	Negative	[40]
CPR1	F-box protein	Protein stability	Negative	[41]
MOS5	E1 ubiquitin activating enzyme	Protein stability	Negative	[71]
MOS2	G-patch and KOW domain containing nuclear protein	RNA-binding	Negative	[72]
SGT1	Chaperone complex	Protein stability	Negative	[40]
MUSE13	TRAFasome	Protein stability	Negative	[36]
MUSE14	TRAFasome	Protein stability	Negative	[36]
MUSE8	AAA-ATPase orthologs of yeast Cdc48	Protein stability	Negative	[46]

BON1, BONZAI1; BAP1, BON1-ASSOCIATED PROTEIN 1; BCAS2, BREAST CANCER-AMPLIFIED SEQUENCE 2; MOS, MODIFIER OF SNC1; SRFR1, SUPPRESSOR OF RPS4-RLD 1.

Recent evidence demonstrates that PIF4 negatively modulates plant defense responses under high temperature. In Arabidopsis, the interaction of PhyB with PIF4 is inhibited at high temperature due to heat-induced inactivation of PhyB. Under high temperature conditions, PIF4 represses defense-related gene expression, thereby increasing plant susceptibility to *Pseudomonas syringae* [15,16•] (Figure 1). High-temperature induced suppression of disease resistance is also observed in *snc1* mutants, including *snc1-1* (which constitutively expresses SNC1 protein) and the *snc1-1 pif4-101* double mutant. However, the level of suppression is more pronounced in *snc1-1* than in *snc1-1 pif4-101*, suggesting that PIF4 plays a negative role in regulating defense responses at high temperature [15,16•]. Furthermore, in *pif4* mutants, several defense-related genes are significantly upregulated, while expression of growth-related genes are significantly downregulated. By contrast, in PIF4-overexpressing plants, enhanced expression of growth-related genes and reduced expression of defense-related genes correlates with enhanced susceptibility to *P. syringae* [16•]. These results further substantiate a role for PIF4 in regulation of the tradeoff between plant growth and defense under high temperatures.

Previous studies showed that PIF4 also indirectly modulates the tradeoff between plant growth and defense in the dark by promoting the activity of the bHLH transcription factor HOMOLOG OF BEE2 INTERACTING WITH IBH 1 (HBI1), which promotes plant growth while suppressing defense signaling [17,18]. HBI1 activity is inhibited upon its interaction with ILI1 BINDING BHLH 1 (IBH1) [18]. In darkness, PIF4 directly interacts with the brassinosteroid (BR)-activated bHLH transcription factor BRASSINAZOLE RESISTANT 1 (BZR1), and the PIF4–BZR1 complex activates the expression of *PACLOBUTRAZOL RESISTANCE* (*PRE*) genes encoding non-DNA binding HLH transcription factors [19,20]. PREs interact with IBH1 to inhibit its activity, and thus promote HBI1 activity, leading to promotion of growth and repression of defense responses (Figure 1).

In contrast to these results, a recent study found no evidence for a PIF4 role in plant defense responses [21•]. In line with this, Arabidopsis wild-type and *pif1 pif3 pif4 pif5* quadruple mutant (*pifq*) plants infiltrated with *P. syringae* showed no significant difference in susceptibility at high temperature (30°C) [21•]. Furthermore, no significant difference in disease susceptibility was observed between wild-type plants and temperature-stable PhyB transgenic lines (35S::PHYB^{Y276H}) at low (23°C) or high (30°C) temperature, suggesting that the phyB/PIF pathway is not primarily responsible for the enhanced susceptibility of Arabidopsis to *P. syringae* at high temperature [21•]. A significant increase in translocation of *P. syringae* effectors, a lack of induction of *ISOCHORISMATE SYNTHASE1* (*ICS1*) and *PATHOGENESIS-RELATED PROTEIN 1* (*PR1*) gene expression, and a lack of accumulation of total SA was observed in plants kept at 30°C compared to plants kept at 23°C [21•]. These observations lead to the conclusion that the increased susceptibility of Arabidopsis to *P. syringae* at high temperature (30°C) is associated with both enhanced pathogen effector translocation and the loss of ICS1-mediated salicylic acid (SA) biosynthesis rather than PIF-modulated susceptibility [21•]. Thus, conflicting views of the roles for PIF4 in the modulation of immune responses are emerging [15,21•].

These contrasting findings might have arisen in part from the different experimental conditions or methods employed [15,21•]. In fact, although they used plants of the same growth stage, these two experiments [15,21•] differed in several respects including inoculation method and inoculum size, growth temperature, light and photoperiod, and relative humidity. The contradictory observations might be primarily caused by the differences in inoculum densities and/or mode of infiltration (spray inoculation with A_{600} 0.002 [21•] versus syringe-infiltration with A_{600} 0.001 [15]). Indeed, significant differences in resistance to *P. syringae* were clearly evident when two different inoculum sizes were used [15]. *pif4-101* and *pifq* mutant plants showed increased resistance compared to wild-type plants when *P. syringae* was spray inoculated with a lower inoculum (A_{600} 0.002); by contrast, no significant differences in susceptibility were observed between wild-type and *pif4-101* mutant plants when a higher inoculum (A_{600} 0.02) was applied [15], similar to the results obtained when plants syringe-infiltrated with an inoculum density of A_{600} 0.001 [21•].

DE-ETIOLATED 1 (DET1) and COP1 (CONSTITUTIVE PHOTOMORPHOGENIC1), a RING-type ubiquitin E3 ligase play important roles in regulating thermomorphogenesis by modulating the stability of key transcription factors, including PIF4, HY5 (ELONGATED HYPOCOTYL5), and HFR1 (LONG HYPOCOTYL IN FAR-RED1) (see Ref. [14]). A recent work highlights the role of PIF4 in the tradeoff between temperature and plant immune signaling through the action of the DET1 and COP1 complex, which negatively affects immunity while promoting growth [15]. In line with this, significantly enhanced resistance to *P. syringae* in *cop1-4*, *cop1-6*, and *det1-1* mutants is associated with markedly reduced PIF4 levels and significantly enhanced expression of defense-related genes, such as *PR1*, *avrPphB SUSCEPTIBLE 3* (*PBS3*), and *PAD4* compared with wild-type plants, particularly in short-day conditions, suggesting that DET1/COP1 signaling negatively regulates defense gene expression through stabilizing PIF4 protein levels [15]. Furthermore, an enhanced *snc1-1* phenotype is observed in *snc1-1 det1-1* and *snc1-1 cop1-4* mutants even at higher temperatures (27°C) [15]. These results thus highlight the importance of

the DET1/COP1–PIF4 signaling module in negatively regulating plant defense.

PIF4 acts downstream of the SUMO (small ubiquitin-like modifier) E3 ligase SIZ1, a key regulator of multiple cellular processes [22,23•]. Under normal conditions (22°C), SIZ1 negatively regulates plant immunity through the sumoylation and activation of COP1 and the stabilization of PIF4 and HY5 levels [23•]. Accordingly, *siz1* exhibits enhanced SNC1-mediated resistance to *P. syringae* at both low and high temperatures (22°C and 28°C) [22,23•], whereas overexpression of *SIZ1* partially rescues the autoimmunity of *snc1* by reducing SNC1 protein levels [22]. Interestingly, COP1 was recently shown to positively regulate plant defense responses by stabilizing the NLR proteins HRT (HYPERSENSITIVE RESPONSE [HR] TO *TURNIP CRINKLE VIRUS* [TCV]) and RPM1 (RESISTANCE TO *P. SYRINGAE* PV. *MACULICOLA 1*) [24]. Therefore, it is possible that COP1 also stabilizes SNC1 protein levels through a direct interaction.

The role of chromatin remodeling in temperature-modulated defense responses

The SWR1 complex (SWR1c) is a conserved Swi2/Snf2-related ATP-dependent chromatin remodeling protein complex that mediates the replacement of histone H2A (HTA) with the H2A.Z variant in euchromatin [25,26]. Increasing evidence indicates diverse roles for SWR1c components and H2A.Z in plant physiological process including plant defense signaling. Previous results demonstrated that mutations in SWR1c components including PHOTOPERIOD-INDEPENDENT EARLY FLOWERING1 (PIE1), SWR1 COMPLEX6 (SWC6), and ACTIN-RELATED PROTEIN6 (ARP6) or H2A.Z lead to constitutive defense responses [26,27]. In line with this observation, the Arabidopsis *arp6* and *hta9hta11* mutants, which show a deficiency in the incorporation of H2A.Z into nucleosomes, exhibit developmental and morphological phenotypes similar those of plants grown at high temperature. These mutants display constitutive expression of warm-temperature-responsive genes and enhanced resistance to *P. syringae* through increased PTI responses [28,29].

These observations point to a possible link between temperature-modulated immune responses and transcriptional reprogramming (Figure 1). Intriguingly, the suppressed ETI responses in *arp6* and *hta9hta11* also indicate that H2A.Z-containing nucleosomes play different roles in modulating PTI and ETI [29]. Although the SWR1c components and H2A.Z proteins are not directly involved in plant defense signaling pathways, they exert their functions through transcriptional gene regulation. However, their specialized functions in PTI and ETI responses have yet to be elucidated. In addition to the roles of the SWR1c complex and H2A.Z-containing nucleosomes in temperature perception and the modulation of plant immunity, other histone-modifying enzymes and chromatin-remodeling complexes are also involved in plant defense responses [30]. Therefore, more in-depth studies are needed to elucidate how these components modulate ETI and PTI responses.

A recent transcriptomic analysis [31•] suggested that at high temperatures, heat shock factors (HSFs), specifically HSFA1, play key roles in the rapid eviction of H2A.Z-containing nucleosomes from temperature-sensitive target genes, thereby facilitating the induction of downstream stress-responsive transcriptional regulators [31•]. It would be interesting to investigate whether the high temperature-dependent inhibition of H2A.Z accumulation in nucleosomes increases the accessibility of target genes for the master regulators involved in plant defense responses.

Temperature-dependent stabilization of NLR proteins

The stability of NLR proteins is strictly regulated by a highly conserved chaperone complex consisting of HSP90 (HEAT SHOCK PROTEIN 90), SGT1 (SUPPRESSOR OF G-TWO ALLELE OF SKP1), and RAR1 (REQUIRED FOR MLA12 RESISTANCE) [32,33]. The ubiquitin proteasome system (UPS) also contributes to NLR protein stability, thereby affecting the activation of these proteins during plant–pathogen interactions [34–38]. 26S proteasome-mediated protein degradation, the most common mechanism for protein stabilization in plants, involves the ubiquitination of substrate proteins through the action of a series of cascade reactions involving E1, E2, E3, and/or E4 ubiquitin ligases [39]. The role of E3 ligase complexes, particularly the SCF (SKP1-CULLIN1-F-BOX) E3 ligase complex, in the temperature-induced modulation of defense responses is well documented. For instance, loss-of-function mutations in the F-box protein CPR1 (CONSTITUTIVE EXPRESSOR OF PR GENES 1) lead to the SNC1-mediated constitutive activation of defense responses at lower temperatures [40,41]. By contrast, silencing of the F-box protein gene, *ACIF1* (*Avr9/Cf-9-INDUCED F-BOX1*), suppresses resistance to *Tobacco mosaic virus* (TMV) and *P. syringae* pv. *tabaci* in tobacco and to *Cladosporium fulvum* in tomato [42] indicating a complex regulation of SCF E3 ligases in plant defense mechanisms.

More recently, the U-box type plasma membrane-associated E3 ligase SAUL1 (SENESCENCE-ASSOCIATED E3 UBIQUITIN LIGASE1) was found to be involved in temperature-dependent resistance mediated by the NLR protein SOC3 (SUPPRESSORS OF CHS1-2, 3) [43•,44]. Loss-of-function mutations of *SAUL1* lead to the activation PAD4-dependent and EDS1-dependent SOC3-mediated autoimmunity at temperatures below 25°C,

despite a moderate increase in temperature-induced suppression of autoimmune responses [44]. Intriguingly, either the loss or overexpression of *SAUL1* leads to SOC3-mediated immunity. SAUL1 does not appear to play a major role in ETI responses, but functions as a positive regulator of PTI and as a virulence target for a yet unknown effector [43•]. SAUL1 might modulate PTI through ubiquitination of one or more membrane-localized negative regulators. Modifications of SAUL1 protein are likely monitored by the NLR immune receptor, SOC3, through an indirect association. It will be critical to identify the effectors recognized by SOC3 and the ubiquitination targets of SAUL1 to fully understand SAUL1/SOC3-mediated PTI responses [43•].

MUTANT *SNC1*-ENHANCING (MUSE) proteins, such as MUSE3, MUSE8, MUSE13, and MUSE14, were recently shown to play important roles in the turnover of NLR proteins by negatively regulating their accumulation through the UPS [36,45,46]. *MUSE8* encodes AtCDC48A, which shares functional similarity with Cdc48, an AAA-ATPase ortholog in yeast [46]. The loss-of-function Arabidopsis mutant *muse8* exhibits enhanced SNC1-mediated immune responses, and exogenous expression of MUSE8 suppresses the temperature sensitivity of yeast *cdc48* mutants [46], indicating that MUSE8 functions in temperature sensitivity. MUSE8 interacts with MUSE3, an E4 ligase involved in the polyubiquitination and proteasomal degradation of SNC1 [45,46], suggesting that MUSE8 plays a role in stabilizing SCN1 levels [46]. Another study demonstrated that the TRAF (tumor necrosis factor receptor-associated factor) domain-containing proteins MUSE13 and MUSE14 interact with the SCF E3 ligase (SCF^{CPR1}) complex to form a TRAFasome that regulates the ubiquitination and subsequent degradation of NLR proteins including SNC1 and RPS2 (*RESISTANCE TO PSEUDOMONAS SYRINGAE2*) to maintain their homeostasis [36]. At low temperatures, the *muse13-2 muse14-1* double mutant displays enhanced resistance and autoimmunity, along with increased accumulation of NLR proteins [36], suggesting that TRAFasomes negatively regulate NLR activity in a temperature-dependent manner. Overall, these studies emphasize the important role of the UPS as a major regulatory node in the tradeoff between plant growth and immunity.

Crosstalk between temperature and hormonal signaling during plant defense responses

Plant hormones play central roles in the outcome of plant defense responses. In particular, the roles of SA, abscisic acid (ABA), jasmonic acid (JA), and ethylene (ET) in regulating plant defense responses are well established (see Refs. [47,48]). ABA positively regulates defense responses during pre-invasion by restricting pathogen entry through stomata but negatively regulates defense responses during post-invasion through its inhibitory effects on SA biosynthesis and SA signaling [49,50]. Moreover, many NLR-mediated defense responses are inhibited at high temperature due to enhanced ABA accumulation and the subsequent inhibition of the nuclear localization of NLR proteins including SNC1 and RPS4 [4,51,52]. For instance, the ABA-deficient Arabidopsis mutant *aba2* exhibits increased resistance against *P. syringae* pv. *tomato* at high temperatures due to the enhanced nuclear accumulation of SNC1 and RPS4. However, the effect of ABA on the nuclear localization of NLRs appears to be independent of SA [4]. Nevertheless, some NLR proteins, such as RPS2 and RPM1, which are localized to the plasma membrane, are not affected by ABA deficiency or temperature stress [4,53,54].

Recent studies have highlighted the effects of high temperature on SA/JA-mediated defense responses [21•,55,56•]. At elevated temperatures, ABA biosynthesis and signaling genes are upregulated and SA-responsive genes are downregulated in susceptible rice plants infected with *Xanthomonas oryzae* [55]. However, rice plants carrying the *Xa7* gene exhibit resistance to this pathogen, even at high temperature, despite the downregulation of SA-responsive gene expression, suggesting that SA-independent defense signaling occurs at high temperature [55]. Identification and cloning of the *Xa7* candidate gene would provide more insights into SA-independent resistance signaling. Similarly, high temperature enhances the susceptibility of Arabidopsis to *P. syringae* by inhibiting SA-biosynthetic and SA-responsive gene expression and upregulating genes involved in JA-mediated signaling and ABA biosynthesis, suggesting there is interplay between SA and ABA/JA signaling [21•].

A recent study [56•] uncovered the role of JA in suppressing SA-mediated defense responses at high temperature in Arabidopsis. PHYTOALEXIN DEFICIENT 4 (PAD4) is required for SA biosynthesis. PAD4-regulated SA-mediated defense responses are generally suppressed at high temperatures. Under these conditions, JA suppresses the expression of *PAD4* and *EDS5* (*ENHANCED DISEASE SUSCEPTIBILITY 5*) [56•]. Intriguingly, JA confers enhanced SA accumulation and defense responses in the absence of *PAD4* through an incoherent-type feed-forward loop by enhancing *EDS5* transcript accumulation [56•]. Therefore, crosstalk between JA and SA plays an important role in alleviating the negative effects of SA on plant growth at high temperature. By contrast, low temperature enhances SA accumulation and the expression of SA-signaling genes, which is associated with enhanced resistance to *P. syringae* infection in Arabidopsis [57••]. Calmodulin-binding transcription activator (CAMTA) transcription factors are thought to regulate cold temperature-dependent expression of SA biosynthesis and responsive genes [57••,58]. Compared to our knowledge of the mechanisms underlying the effects of temperature on ABA-dependent and SA-dependent defense responses, little is known about its effects on defense responses mediated by JA and ET. Further

research on this topic should increase our understanding of plant defense responses against necrotrophic pathogens and pests.

Conclusions

The outcomes of plant–pathogen interactions are affected by temperature. Global climate change is expected to pose a great challenge to crop production. A comprehensive understanding of the consequences of temperature fluctuations to plant defense responses at the molecular level would be of great epidemiological importance. Considerable progress has been made in understanding the genetic and molecular mechanisms underlying the temperature sensitivity of *R* genes. Future studies should focus on the epigenetic changes, including histone modifications and conformational changes in chromatin architecture, associated with the effects of temperature on immune responses. SCF complex proteins play important roles in plant defense responses against both pathogens and abiotic stress, as an integral part of the protein degradation machinery. Studies aimed at identifying novel SCF complex components and exploring the direct interplay between pathogens and interactors of the host plant's UPS associated with temperature stress are needed to better understand how the UPS dynamically mediates defense signaling in plants.

Targeting common master regulators such as SIZ1, COP1, and PIF4 for genetic improvement is a promising approach for developing crops resistant to combined biotic and abiotic stresses. Since our current understanding of the mechanisms underlying the suppression of plant resistance at high temperature is largely based on model species, a key challenge will be to apply this knowledge to economically important crop plants in order to develop temperature-insensitive, disease-resistant plants with increased productivity under aberrant climatic conditions.

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- • of special interest
- •• of outstanding interest

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